

SPINAL STENOSIS

A SUMMARY AND REVIEW

BRUCE F. WALKER D. C. *

Abstract: A review of the etiology, clinical, radiological and laboratory presentation, differential diagnosis and management of spinal stenosis is presented.

Index Terms: Spinal stenosis, central stenosis, lateral recess stenosis, back pain, chiropractic.

Summary: The shape and size of the lumbar vertebral canal governs the amount of space available for nerves that the canal transmits, and if this space is reduced by encroachment of the boundaries of the canal the anatomical anomaly is referred to as spinal stenosis (1), a term coined by Verbeist (2).

Spinal stenosis can occur in the central canal, lateral recess or intervertebral foramen (3). It can also be considered anatomically as being either lateral (apophyseal), giving rise to compression of the emerging nerve roots, or midline (laminar), resulting in compression of the cauda equina or the thecal sac (7). Bony compression of the nerve roots may arise as a result of sub articular entrapment, pedicular kinking or foraminal impingement due to posterior joint subluxation (1)(7).

Stenosis can be **congenital** in nature and if so may not by itself cause nerve compression. It only renders the patient more likely to nerve compression (1). It may be acquired from the degenerative process with its attendant spurs and bony encroachment, disc or ligament encroachment (particularly ligamentum flavum) (1)(4). Other causes are found under "Spinal stenosis as a complication".

Combinations of causes may be involved in spinal stenosis eg: a congenitally narrow canal with surrounding degeneration (4).

Stenosis is more common with advancing age (older than 65) this is a result of advancing degenerative changes (5). Stenosis is encountered mainly at the

L4/5 level but also occurs at the L3/4, L5/S1 and other levels (6). Many people have multi-segmental stenosis and indeed may not have associated symptoms (7). Symptoms when present include backache and sciatic pain, sciatic pain only, paresthesias and progressive weakness. When radicular pain is present it implies the stenosis is originating from the lateral recess (7). This leg pain often worsens with walking (5) and is relieved by stooping, sitting or lying down in a flexed position (so-called neurogenic claudication). This differs from vascular insufficiency (or claudication) in that with stenosis standing does not relieve the pain (7)(9).

Occasionally with severe stenosis autonomic symptoms may occur. Ram et al reported a case of a patient who developed intermittent priapism on walking caused by stenosis. This was ameliorated by surgical decompression (8).

The confusing aspect of symptom presentation is that patients may have bilateral lateral recess stenosis but mono-radicular pain (7). Porter speculates that symptoms are the result of inadequate oxygenation of the cauda equina arising from arterial ischaemia or venous engorgement, which permits adequate nerve function at rest but inadequate function during exercise. (9).

Differential Diagnosis:

The differential diagnosis of spinal stenosis must include all causes of low back pain and radicular pain. The most common of these are (10)(11)(12):

1. Disc bulges, herniation or sequestration.
2. Facet joint dysfunction.
3. Extra foraminal nerve root compromise.
4. Myofascial trigger points.

The prudent clinician must also exclude neoplasia including retro-peritoneal tumours, infection, vascular disease (including aortic aneurysm) and renal lesions (3)(5).

Customary Examination Procedures:

1. Physical/Orthopaedic Examination:

It must be remembered that co-existence of lumbar stenosis with either a herniated disc or spondylosis is very common (4). Examination may therefore reveal symptoms of one or all co-existing conditions.

Stenosis in isolation will demonstrate a straight leg raising that is often less painful than true sciatica (5). Forward bending often relieves the pain in stenosis (13) and not in true sciatica. Neurological examination is often unremarkable with stenosis except in advanced cases. (5).

Importantly signs indicative of stenosis may be more evident in patients after activity (3).

2. Diagnostic Imaging:

- (a) Plain radiography.
Yochum and Rowe describe a number of measurements which can be used to assess spinal stenosis (14). These include
 - (i) Macnab's Line and Hadley's "S" curve can detect facet imbrication or subluxation (14). Bony root entrapment syndromes are frequently associated with subluxation of the posterior facets often to a marked degree (7).
 - (ii) Interpediculate distance which is measured on an antero-posterior projection (14).
 - (iii) Eisenstein's Method for sagittal canal measurement. This appears to be the most reliable of the plain radiograph measurements for sagittal canal diameter (15).
 - (iv) Canal/Body Ratio. Taken from a lateral view the higher the ratio the smaller the spinal canal (14).

Yochum and Rowe conceded that computerised tomography must be

performed to make a definitive diagnosis of stenosis (14).

- (b) Computerised Tomography (CT).
With the advent of high resolution CT it is possible to image the cross sectional anatomy of the spinal canal and demonstrate various pathological processes involving the articular facets and the laminae arches with great precision (16).

In a study of 122 patients Bell et al compared CT with myelography in the diagnosis of spinal stenosis. Myelography was only slightly more accurate than CT (93% Vs 89%). (17).

- (c) Myelography.
Metrizamide myelography is highly accurate in the diagnosis of spinal stenosis (17). It gives the added advantage of visualising the thoracolumbar junction and affords the opportunity to diagnose occult tumours (17).
- (d) Magnetic Resonance Imaging (MRI).
High resolution MR images match myelography in the capacity to image the spinal canal contents. Additionally, the contents of the canal can be differentiated from one another (18).
- (e) Ultrasonography.
Ultrasound of the spine for the purposes of detecting stenosis is not a reliable method but may have use in the area of screening (19)(20).

Other Laboratory Tests:

Nerve conduction studies and EMG.

Johnsson et al state that in cases with suspected or verified spinal stenosis, a bilateral EMG and neurographic investigation gives valuable information about the degree of neurogenic affection and excludes polyneuropathy (21).

Goals of Treatment:

- 1. Reduce pain and inflammation.
- 2. Decrease neural and vascular impingement.
- 3. Improve function.
- 4. Retard progression.

Chiropractic management:

Rosomoff advocates a non-surgical aggressive treatment regime for lumbar spinal stenosis (22). He suggests 3-4 weeks of intensive management with an initial in-patient program moving to out-patient status with improvement.

The treatment program includes physical therapeutic modalities, trigger point injections, muscle stretching and strengthening, range of motion work, gait training and postural realignment. He advocates occupational therapy, nutritional counselling and where necessary behavioural therapy (22).

Kirkaldy-Willis states that in patients with dynamic recurrent lateral stenosis, adjustment (manipulation) of the spine into flexion and axial rotation in the pain free direction opens up the canal and foramen and may be of benefit. With fixed gross stenosis he is more cautious about manipulation (23). In central stenosis Kirkaldy-Willis suggest that manipulative adjustments are rarely indicated but may have a role (23).

Acupuncture may be useful in the management of the pain of stenosis (24). Steriodal and non-steroidal agents may be useful where inflammation is involved (22). Some advocate a trial of anti-inflammatories over several months to reduce neural and soft tissue inflammation to allow recovery, however their utility in the chronic phase has not been clearly established (28).

Surgical decompression with or without fusion may be the only answer in intractable cases (3).

Prognosis:

According to Rosomoff (22) it is possible even in the most severely disabled to return such individuals to full functional capacity with a multi-disciplinary therapeutic program. After extensive decompression laminectomy, there is a risk of post-operative instability where fusion is not performed (25). However, Weinstein et al suggested in a study that patients who received surgical decompression, obtained definite measurable benefit during a 1-3 year period following surgery (26).

Spinal Stenosis as a Complication:

Apart from idiopathic congenital cases lumbar spinal stenosis may be caused by:

1. Achondroplasia
2. Pagets disease
3. Fluorosis
4. Retrolisthesis
5. Spondylolisthesis
6. Degeneration with or without subluxation
7. Post-traumatic
8. Post-operative
9. Disc encroachment or sequestration
10. Ligamentum flavum hypertrophy
11. Facet joint capsule hypertrophy
(1,3,4,5,6,7,9,13,27)

References:

1. Bogduk N., Twomey L., Clinical Anatomy of the Lumbar Spine. Churchill Livingstone. Melbourne. 1987. pp.46-48.
2. Verbeist H. Sur certains formes rares de compression de la queue de cheval. Hommage 'a Clovis Vincent. Paris: Maloine, 1949. pp.161-174.
3. McCulloch J.A. Spinal Stenosis. In:Spine Update 1987. Ed. Genant H.K. Raiology and Research Foundation. California. pp. 191-202.
4. White A.A., Panjabi M.M. Clinical Biomechanics of the Spine. J.B. Lippincott Co. 1978. pp. 292-293.
5. Frymoyer J.W., Gordon S.L. (Eds). New Perspectives on Low Back Pain. American Academy of Orthopedic Surgeons. 1988. pp. 50-51.
6. Kirkaldy-Willis WIH. Managing Low Back pain. Churchill Livingstone. 1983. pp. 33-43.
7. Macnab I. The Pathogenesis of Spinal Stenosis. In:Spinal Stenosis: State of the Art Review, Hopp E. (Ed). Spine. Hanley and Belfus. 1987. pp. 269-381.
8. Ram Z. et al. Intermittent Priapism in Spinal Canal Stenosis. Spine: Vol 12. No. 4. 1984. pp 377-378.
9. Porter R.W. Management of Back Pain. Churchill Livingstone. 1986. pp. 110-121.
10. Rauschnig W. Normal and Pathologic Anatomy of the Lumbar Root Canals. Spine. Vol. 12. No. 10. 1987. pp. 1008-1019.
11. Bogduk N., Twomey L.t., Clinical Anatomy of the Lumbar Spine. Churchill Livingstone. Melbourne. 1987. p.139.
12. Simons D.G., Travell J.G. Myofascial Origins of Low Back Pain. Post Graduate Medicine. Vol. 73. Feb. 1983, p. 66.
13. Finneson B.E. Low Back Pain. Second Edition. 1980. J.B. Lippincott. pp. 427-434.
14. Yochum T.R.Y., Rowe L.J. Essentials of Skeletal Raidology. Vol. 1. Williams and Wilkins. 1987. pp. 191-198.

15. Eisenstein S. Measurements in the Lumbar Spinal Canal in Two Racial Groups. Clin. Orthop. Rel. Res. 115:43,1976.
16. Ciric I et al. The Lateral Recess Syndrome. J. Neurosurg. 53:433-443, 1980.
17. Bell G.R. et al. A Study of Computer Assisted Tomography.II. Spine. Vol. 9. No. 6. 1984. pp. 552-556.
18. Yochum T.R.Y., Rowe L.J. Essentials of Skeletal Radiology. Vol. 1. Williams and Wilkins. 1987. p. 287.
19. Tervonen O., Koivukangas J. Transabdominal Ultrasound Measurement of the Lumbar Spinal Canal. Spine. Vol. 14. No. 2. 1989. pp.232-235.
20. Legg S.J., Gibbs V. Measurement of the Lumbar Spinal Canal by Echo Ultrasound. Spine. Vol. 9. No. 1. 1984. pp.79-82.
21. Johnsson K., Rosen I., Uden A. Neurophysiological Investigation of Patients with Spinal Stenosis. Spine. Vol. 12. No. 5. 1985. pp. 483-487.
22. Rosomoff H.L., Rosomoff R.S. Non Surgical Aggressive Treatment of Lumbar Spinal Stenosis. In:Spinal Stenosis. Spine: State of the Art Reviews. Hanley and Belfus Inc. 1987. pp. 383-400.
23. Kirkaldy-Willis W.H. Managing Low Back pain. Churchill Livingstone. 1983. p. 180.
24. Jaskoviak P. Applied Physiotherapy. Amer. Chiro. Assn. 1986. pp. 81-82.
25. Johnsson K.E., et al. Post-operative Instability after Decompression for Lumbar Spinal Stenosis. Spine. Vol. 11. No. 2. pp. 107-110. 1986.
26. Weinstein J.M., Scafuri R.L., McNeill T.W. The Rush-Presbyterian St. Lukes Lumbar Spine Analysis Form:A Prospective Study of Patients, with Spinal Stenosis. Spine. Vol. 8. No. 8. 1983. pp. 891-896.
27. Frymoyer J.W., Gordon S.L. (Eds). New Perspectives on Low Back Pain. American Academy of Orthopedic Surgeons. 1988 p. 232.
28. LeBlanc F.E. (Ed). Scientific Approach to the Assessment and Management of Activity-Related Spinal Disorders. Spine. Vol. 12. No. 7S. 1987. p.S24.

